THE VALUE OF TRACE MINERALS FOR CATTLE GRAZING BLUESTEM GRASS

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by

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INTRODUCTION

Inorganic elements are required by all forms of life for normal growth and reproduction. The actual role played by many of these mineral elements in the body is not fully understood. Considerable research has been conducted in recent years in an attempt to more fully understand the nutritive role of mineral elements.

Today there are 15 mineral elements considered essential for life. These mineral elements can be divided into two classes, depending upon the amount of each needed by the body. Those that are required in substantial quantities are called the macro or major minerals and include calcium, phosphorous, sodium, potassium, chlorine, magnesium, and sulfur. Those required in minute quantities are termed trace mineral elements and include iron, iodine, manganese, molybdenum, copper, cobalt, zinc, and selenium.

The literature contains much conflicting information as to the exact requirements of these trace minerals needed by the bovine.

This report was prepared in an attempt to summarize results obtained from experiments conducted by others concerning the trace mineral requirements of cattle. An attempt was also made to try to determine if cattle grazing bluestem grass would be deficient in any of these trace mineral elements.

ZINC

Zinc is undoubtedly a dietary essential to the bovine. This has been shown by Ott *et al.*, (1965) and Miller *et al.*, (1963). According to Maynard and Loosli (1962), the zinc content of the body is approximately three mg per cent. The highest concentrations of zinc are found in the epidermal tissues, such as the skin and hair, but traces also are found in the bones, muscles, blood, and various organs. Mammals secrete zinc in milk and in a much stronger concentration in colostrum.

Maynard and Loosli (1962) reported that zinc functions with the respiratory enzyme carbonic anhydrase, which is found in the red blood cells and elsewhere in the body. Carbonic anhydrase plays an essential role in eliminating carbon dioxide and has been found to contain 0.3 per cent zinc. This mineral also was found as a part of the enzyme, uricase, and as a part of crystalline insulin as reported by Dukes (1955).

According to the National Research Council (1963) the requirement for zinc by beef cattle has not been established. Recent research has shown that cattle on certain types of rations may respond to supplemental zinc. Ott (1965) did work to investigate the effect of zinc supplementation on calves fed a low zinc diet. The 7 to 11 week old calves were divided into a control basal diet group which received three mg of zinc per kg of diet; the other group received 100 mg of zinc per kg of diet per day. In the basal diet group, zinc deficiency symptoms began to appear within three weeks. These symptoms included

swollen feet with open scaly lesions followed by alopecia and general dermatitis which was most severe on the legs, neck, head, and around the nostrils. The deficiency was corrected in three to four weeks by feeding the basal diet plus 100 mg of zinc per kg diet. When the animals were placed on the basal diet after receiving the zinc supplement for 10 weeks, the deficiency symptoms developed within three weeks. It would thus appear that large amounts of zinc are not stored for long periods of time by the animal body. Work by Miller et al., (1963) indicated that calves fed a diet containing 3.6 p.p.m. of zinc developed severe parakeratosis. The zinc deficiency symptoms were similar to those found by Ott (1965) and included: inflammation of the nose and mouth with submucosal hemorrhages; unthrifty appearance; rough hair coat; stiffness of the joints with soft swelling of the feet in front of the fetlocks; breaks in the skin around the hoofs that later became deep fissures; dry scaly skin on the ears; thickening and cracking of skin around the nostrils; appearance of horny outgrowths of the mucosa on the lips and dental pads; frequent gnashing of teeth; alopecia starting on the rear legs; red, scabby, and shrunken skin on the scrotum; and bowing of the rear legs. Zinc content of the blood, pancreas, liver, and other organs, and of the bones and teeth was lower for deficient animals than for zinc-supplemented animals. Carbonic anhydrase activity of the blood also declined. These workers found that supplementing the deficient diet with 43 p.p.m. of zinc prevented these symptoms from developing and

affected rapid cures of deficient animals.

Miller et al., (1963) conducted additional studies with zinc. Holstein bull calves were fed a semipractical diet which contained 8.6 p.p.m. of zinc. This ration was fed until 29 weeks of age. They were then fed the same diet with additional amounts of zinc for eight weeks. They again checked blood carbonic anhydrase activity, a necessary enzyme in red blood cells, and also checked weight gains, feed consumption, testicle size and weight, testicle zinc content, and blood zinc content. They found that the calves on the basal diet of 8.6 p.p.m. of zinc performed as well as those fed the additional zinc.

Miller et al., (1964) did work with goats given a diet containing 5.3 p.p.m. of zinc. This experiment resulted in the same deficiency symptoms as reported for calves except for the testicles of the goats which were reported as being extremely small. Microscopic examination of the testicles from the zinc deficient goats were in agreement with those reported earlier for rats. Pitts et al., (1966) conducted work to determine the effect of a zinc deficiency from two to five months of age on reproduction in Holstein bulls. This work was in contrast to previous experiments, and the reproductive characteristics measured were not significantly affected.

Miller et al., (1966) attempted to determine the influence of a zinc deficiency on dry matter digestibility in ruminants. When zinc-deficient calves were fed at the same level of intake as normal animals, they grew at a much slower rate and were less

efficient in converting feed to body weight gain. Miller also found that dry matter digestibilities were not affected by the zinc deficiencies. From this they concluded that the reduced feed efficiency in zinc-deficient animals was caused by a reduced utilisation of digested nutrients. Previously Miller and Cragle (1965) conducted research to determine the site of absorption and endogenous secretion of zinc in cattle and found that zinc was absorbed from the abomasum and lower small intestine while secretion of zinc took place in the upper small intestine.

Miller et al., (1965) experimented to determine the effect of zinc deficiency on wound healing in cattle. They found that large areas around surgically imposed wounds developed parakeratosis in zinc-deficient calves. They also found that a zinc deficiency retarded the rate of new skin growth following surgical removal of the skin.

Haaraven (1963) did work concerning the zinc requirement of cattle for the prevention of itch and hair licking at different calcium levels in the feed. He found that itch and hair appeared mainly in cows receiving less than 45 p.p.m. of zinc in normal calcium feeding. He calculated that for every calcium increase of 0.1 per cent above 0.3 per cent in dry matter that the corresponding zinc increase should be 16 p.p.m. In addition, many groups of workers have found that an excess of calcium in the diet ties up some of the available zinc.

Legg and Beare (1960) reported zinc deficiencies of cattle

grazing on the Bebice Savannahs. The more severe cases resulted in parakeratosis over about 40 per cent of the body surface. The following areas of the body were found to be affected, usually in the order given: muzzle, (except when black) vulva, anus, top of tail, ears, back of hind limbs, knee fold, flanks and neck. This work was done in British Guiana where the zinc deficiencies occurred from March through June. In 1959, 20 cattle out of 1000 showed symptoms of a zinc deficiency. They found the over-all body condition of cows affected to be generally below average; yearlings showed little or no live weight gains for some months, whereas young calves were otherwise normal. When the affected animals were given zinc sulphate either orally (two gms per week) or by injection (one gm per week), new hair growth was evident one week after treatment started; the animals appeared normal after three weeks of treatment.

Underwood (1962) working with information about zinc-deficient British Guiana cattle, stated that the minimum zinc requirement of cattle had not yet been given with any degree of accuracy. In the zinc-deficient British Guiana area the levels of zinc in the three principal range grasses of the affected area were found to be 30-42, 26-36, and 18-22 p.p.m. of zinc on the dry basis. However, as the amounts and proportions of these grasses actually consumed was not known, all that could be concluded was that the minimum zinc requirement of these grazing cattle was not less than about 20 p.p.m. and probably was nearer 30 p.p.m. According to Underwood (1962), pasture plants and

forages growing on normal soils usually contain 30-100 p.p.m. of zinc on the dry basis.

Duncan and Epps (1958) tested Pinehill bluestem and Slender bluestem for zinc content at three different stages of maturity. They obtained the following values:

Species and Maturity Stage	Zinc (p.p.m.)
Pinehill bluestem	
Early leaf	12.2
Full leaf	1.0
Mature green	46.8
Slender bluestem	
Early leaf	12.2
Full leaf	8.4
Mature green	12.0

They presumed these amounts of zinc to be sufficient; however they emphasized that the requirement of zinc for cattle had not yet been established.

French et al., (1957) compiled a table in an attempt to summarize knowledge regarding normal levels in forage crops for mineral elements known to be important in animal nutrition. They gave a range for zinc content of grasses on a dry matter basis under three categories; low, normal, and high. A value was listed of 1-5 p.p.m. of zinc for low, 10-30 p.p.m. for normal, and 50 p.p.m. for high zinc content. They felt there was a possible chance that aluminum in the diet protected the animal

against zinc toxicity. However, Underwood (1962) stated that very little is known about zinc toxicity in ruminants.

From the literature it appeared that a zinc deficiency in the Kansas bluestem area was not very apt to occur. However, it was noted that a zinc deficiency might be possible during the rapid growing season of the grass according to the values obtained by Duncan and Eppe (1958) working in Louisiana. Furthermore, it seemed that the cattle grazing on mature green grass received an ample supply of zinc.

MOLYBDENUM

According to Underwood (1962), molybdenum is classified as an essential trace element. An earlier edition by Underwood (1957) classified molybdenum as "probably essential". But it appears from the literature that no one is certain about the essentiality of molybdenum.

Molybdenum has received the greatest amount of attention by cattle raisers from the standpoint of toxic levels rather than deficiency levels. However, Ellis *et al.*, (1958) have shown beneficial response from molybdenum in lambs fed a semi-purified diet containing 0.36 p.p.m. of molybdenum to which molybdate was added to raise the molybdenum content to 2.36 p.p.m. The lambs receiving the ration with added molybdenum made significantly faster gains than the controls in two separate feeding trials. The experimenters concluded that this experiment demonstrated a nutritional role for molybdenum in the growing ruminant in that it is required for optimum cellulose digestion by the rumen microflora. The researchers further suggested that part of the frequently reported beneficial effect of alfalfa ash on ruminants fed poor quality roughage rations was due to the molybdenum content of the ash. Ellis and Pfander (1960), did further research on molybdenum as a possible component of the "alfalfa ash factor for sheep". Three subsequent experiments were conducted to try to determine if molybdenum was the component in alfalfa ash that gave the desired response. These experiments excluded molybdenum per se as the beneficial

component of alfalfa ash in stimulating feed consumption and cellulose digestibility of poor quality roughage rations. Underwood (1962) pointed out that many pastures grazed regularly by sheep and cattle contain lower concentrations of molybdenum than the 0.36 p.p.m. used in the experiment conducted by Ellis and workers.

Another factor attributed to a molybdenum deficiency in sheep is that of renal xanthin calculi. A high incidence of such calculi has occurred in wethers grazed on the "Moutere Hills" of the south island of New Zealand. Underwood (1962) reported that Askew obtained evidence pointing to low molybdenum intake as the cause of the calculi. These pastures were found to contain only 0.03 p.p.m. of molybdenum or less compared with concentrations up to 0.4 p.p.m. molybdenum in "non-calcul" areas nearby. Correspondingly, sheep from the deficient area had subnormal levels of molybdenum in their livers. Later the deficient pastures were treated with lime, which in turn raised the molybdenum levels in the grass. This treatment stopped the development of calculi in sheep. However, protein and other constituents were changed by the liming process, so the xanthin calculi formation may not have been due strictly to a lack of molybdenum.

As mentioned, molybdenum toxicities have received considerably more attention than molybdenum deficiencies. This toxicity is sometimes referred to as molybdenosis. The symptoms are scouring, unthriftiness, and rough hair coat in the earlier

stages. In more advanced stages the hair often changes color as, for example, a red animal changing to a straw color or a black changing to a mouse-gray color. Other advanced-stage symptoms include dehydration, arching of the back, listlessness and weakness, brittle bones, emaciation, and in extreme cases, death results. Spencer (1958) reported that younger animals are affected more severely than older animals. Research indicates that molybdenum consumed by the animal through succulent pasturage is, amount for amount, more harmful than that ingested in cured hay. Spencer et al., (1958) stated that opinions vary as to how much molybdenum is necessary to be toxic to livestock. They felt many factors were involved such as the amount of toxic forage eaten in proportion to other forages, the chemical nature of the non-toxic portion, and whether or not the animal had a steady diet of the toxic forage. They reported that some workers believe the critical point to be around 5-6 p.p.m. of molybdenum in forage (air-dry basis); others placed it around 10 p.p.m., while still other workers set the limit at 15 or 20 p.p.m. Cook et al., (1963), working in Nevada on toxicity of molybdenum, gave the range of 5-30 p.p.m. as the critical level of molybdenum. Duncan and Epps (1958) reported a toxicity of molybdenum called "teartness" developed in cattle fed rations containing 8-12 p. p.m. of molybdenum. They felt that pastures containing two p.p.m. or less were generally considered safe.

Cunningham et al., (1952) did considerable work in trying to overcome molybdenum toxicity in cattle. Cattle were

continually lost due to molybdenum toxicity in the Swan River Valley of Manitoba located in South Central Canada. They found the herbage there contained up to 25.6 p.p.m. of molybdenum. The cattle were treated and the toxicity was overcome by the daily administration of two gms of copper sulphate as either a drench or a salt lick. Cunningham et al., (1952) also produced the disease experimentally in cattle by drenching with ammonium molybdate, but considerably more elemental molybdenum than found in toxic forage was required to produce molybdenum toxicity symptoms in barn-fed cattle. This is in agreement with Spencer et al., (1958) who believed that molybdenum taken into the body through succulent forage is more harmful than the same amount of molybdenum taken into the body in the form of cured hay. Cunningham and workers also obtained evidence to show that the toxicity may pass through the dam into the milk and affect the calf but that cattle soon develop a resistance to the passage of ingested molybdenum into the milk. Also, Cunningham reported that during rainy periods when molybdenum toxicity was most severe, as many as 30 per cent of the calves died as well as some mature stock. These workers reported the symptoms previously mentioned and in addition pointed out that affected animals show extreme weakness of the legs, enlargement of the lower joints, and show extreme difficulty in rising. Hemoglobin content of the blood was found to be lower. They found that scouring stopped suddenly when the cattle were moved to unaffected areas. Scouring began again within one to five days if the animals were returned

to the toxic area.

Cook et al., (1963) conducted a trial to study the effect of large doses of inorganic molybdenum administered daily to steers on grass. Sodium molybdate was administered once daily in capsule form to 24 month old steers. The steers were divided into three groups. Group one was the control, two received 68 mg of molybdenum per 100 pounds body weight, and three received 136 mg of molybdenum. Toxic symptoms developed at 25 days for those receiving molybdenum and the test had to be stopped at 100 days due to the severity of the molybdenum toxicity. At this time the steers receiving molybdenum weighed less, had higher levels of plasma molybdenum and more liver iron. They found that plasma and liver copper did not differ significantly. Possibly this might have suggested that physiologically available copper may have been limited. The treatment did not significantly affect bone composition. Also working to study the effect of large doses of inorganic molybdenum, Lesperance and Bohman (1963) fed inorganic molybdenum to weanling heifers at the rate of 100 p.p.m. of the diet. Plasma, liver, and skeletal analyses showed inorganic molybdenum created severe symptoms of molybdenum toxicity. Plasma molybdenum was a better indication of molybdenum intake than liver molybdenum levels. Interactions of molybdenum end copper were not clear-cut.

Another experiment concerning molybdenum toxicity was conducted by Chapman and Kidder (1963) who experimented with yearling Brahman crossbred Angus heifers grazed on grass at Belle

Glade, Florida. The heifers received an equivalent of 250 mg (65 p.p.m.) of molybdenum per animal per day. The treatment of molybdenum resulted in decreased blood hemoglobin and decreased copper stores and increased iron stores in the liver.

According to Underwood (1962) molybdenum was readily absorbed from the intestinal tract and was excreted mainly in the urine. Sulphate was found to reduce molybdenum retention in the tissues, presumably through increased urinary excretion. Dick (1956) felt a possible explanation for this was that inorganic sulphate interferes with and prevents the transport of molybdenum across membranes. This in turn caused a rise in the sulphate concentration in the kidney, and reabsorption of the molybdenum was then blocked by the inorganic sulphate. Work reveals that molybdenum content of the soil and its pH appear to be the main items in determining the level of this element in plant species.

Duncan and Epps (1958) analyzed Pinehill bluestem and Slender bluestem for molybdenum content at three different stages of maturity. The following values were obtained:

Species and Maturity Stage	Molybdenum (p.p.m.)
Pinehill bluestem	
Early leaf	less than 0.06 p.p.m.
Full leaf	less than 0.06 p.p.m.
Mature green	0.12

Slender blusstem

Narly leaf	less than 0.06 p.p.m.
Full leaf	less than 0.06 p.p.m.
Mature green	less than 0.06 p.p.m.

Glendening et al., (1952) attempted to determine the molybdenum content of big and little bluestem grass near Manhattan, Kansas, and obtained the following values:

Prairie Grass, Mostly Dry Bluestem, Collected in Winter	Molybdenum (p.p.m.)
Average	1.0
Minimum	0.2
Maximum	1.7
Prairie Grass, Mostly Green Bluestem, Collected on Dates Shown	
April 19	2.4
July 17	1.1

The above values are considerably higher than those of Pinehill and Slender blusstem found in Louisiana. However, these values fall in the range generally considered safe for cattle.

Molybdenum seemed to be interrelated with other minerals in the general metabolism of the body. Underwood (1962) reported that high molybdenum intake often is accompanied by interference with phosphorous metabolism giving rise to problems of lameness, joint abnormalities, failure of cows to conceive, and lack of

libido in young bulls. Another interaction that appears to be more important is that between molybdenum and copper. This has been known since the early work of Ferguson (1938) who found that scouring of cattle caused by large intakes of molybdenum could be treated by copper sulphate therapy. The literature at present is not too clear as to the mechanisms involved in this relationship. It appears that the ratio of dietary copper and molybdenum are the principal factors in determining the toxicity of any particular level of molybdenum intake. The teart disease of England, New Zealand, California, and Nevada have been controlled by giving large doses of copper to the suffering animals. In New Zealand a disease of cattle called "peat scours" was reported by Underwood (1962) as often occurring on reclaimed peat land. A similar disease called "falling disease" was reported in Western Australia by Bennetts and Hall (1939). In these areas a moderate excess of molybdenum caused scouring only when the pasture was below normal in copper.

As previously mentioned, sulphate has been found to reduce molybdenum retention and thus reduce the chances of molybdenum toxicity on diets considered possibly dangerous due to high molybdenum levels. Work done by Vanderveen and Keener (1964) contradict this theory. In their experiment Holstein heifers which received diets containing from 5-50 p.p.m. of molybdenum, and no added sulphate sulfur, did not develop any of the symptoms of molybdenum toxicity. Heifers which received 50 p.p.m. molybdenum with 0.3 per cent sulphate sulfur developed alopecia and

achromatrichia; however no other symptoms of molybdenum toxicity were observed. When the ration contained 100 to 200 p.p.m. of molybdenum with 0.3 per cent sulphate sulfur, the animals became emaciated, developed alopecia, achromatrichia, and lost nervous control of their rear quarters within three weeks. No changes in liver copper or blood serum copper were observed when the animals received this high molybdenum diet. When copper was added to the diet of heifers which developed achromatrichia and alopecia, the molybdenum toxicity symptoms were completely corrected.

Goodrich and Tillman (1966) conducted an experiment at Oklahoma State University to study the interrelationship of copper, molybdenum and sulfur in ruminant nutrition. Two levels of each mineral in all combinations were fed to sheep. Levels of 10 and 40 p.p.m. of copper, 2 and 8 p.p.m. of molybdenum and 0.10 and 0.40 per cent of sulfur were fed. Response criteria were growth rate, hematology, and blood and liver mineral levels. They found that increasing sulfur in rations containing two p.p.m. of molybdenum reduced gain and efficiency. This was not found to be true when the diet contained eight p.p.m. of molybdenum. It appeared that higher levels of molybdenum overcame some of the detrimental effects of the 0.40 per cent sulfate level. Hemoglobin concentration and erythrocyte counts were not significantly affected by treatments. The workers found that plasma phosphorous was affected by both copper and sulfur levels in the ration. High levels of sulfur decreased phosphorus

plasma while high levels of copper increased plasma phosphorus. Increasing molybdenum from two-eight p.p.m. caused a significant reduction in plasma phosphorus levels when 0.10 per cent sulfur was fed but had no effect when the diet contained 0.40 per cent sulfur.

The researchers found there was a three-way interrelationship among copper, molybdenum and sulfate. Higher levels of sulfur or molybdenum reduced copper storage in the liver. Increasing the copper in the ration resulted in increased storage of copper in the liver. It appeared that lambs on high molybdenum levels had low liver copper and high plasma molybdenum, while those with high levels of plasma copper seemed also to have high plasma molybdenum levels.

In summary, it appears that an adequate amount of molybdenum is present in bluestem grass the year around; however it is not present in amounts considered dangerous. The possibility of molybdenum toxicity in cattle on bluestem grass in the Manhattan, Kansas, area seems even more remote since native grass in this area is very high in copper content.

COPPER

The presence of copper in plant and animal tissue has been a known fact for many years. Maynard and Loosli (1962) reported that approximately half the copper in the body is found in the muscles, while supplies are found also in the bone marrow, liver, and to a lesser degree elsewhere. According to Underwood (1962) the first real evidence that copper was an essential element developed from studies conducted at Wisconsin in 1924 on hemoglobin regeneration in rats suffering from milk anemia. Later it was learned that copper was needed along with iron in the formation of the hemoglobin molecule. Underwood also stated that several copper protein compounds have been isolated from plant and animal tissues, some of which are ensymes with oxidative functions. Evidence has indicated that tyrosinase, laccase, and ascorbic acid oxidase, cytochrome oxidase, and uricase are copper compounds.

Neal and Becker (1933) first reported a copper deficiency in grazing cattle. This was a natural deficiency in the "salt-sick" cattle of Florida. Soon after this, copper deficiencies were reported in both Holland and Australia. Later it was discovered that the poisoning of sheep grazing in Australia was due to copper toxicity. This was further complicated when it was discovered by Dick (1952) that copper was interrelated with inorganic sulphate and molybdenum.

It was reported by the National Research Council (1963) that copper deficiencies are mainly "area" diseases. They

listed the following as symptoms of a copper deficiency: depraved appetite, loss of condition, stunted growth, rough hair coat, and anemia. The National Research Council (1963) stated that most cases of a copper deficiency start with severe diarrhea. Often the hair coat is not only rough, but also appears to be bleached due to loss of pigment. This bleached appearance has been found to be caused by a copper deficiency which interferes with the synthesis of keratin, the principal constituent of hair and wool. Often the ends of the leg bones above the pasterns become swollen. The bones become weak and ribs and legs are easily broken. Older cattle sometimes develop osteomalacia. Cows suffering from copper deplation may fail to conceive, or if they do conceive often have problems of retained placentas and give birth to calves with congenital rickets. Davis (1951) observed a high percentage of monstrosities in calves from cows grazed in areas deficient in copper. He found as many as ten per cent of the calves are sometimes monstrosities, often found possessing enlarged heads with missing bones and abnormal formation of bones. In severe cases he found that the cows would not cycle and that the bulls became sterile due to destruction of the germinal tissue. Cunningham (1944) reported that falling disease or "szootic ataxia" in Australia was a copper deficiency. This resulted in the animal's staggering, falling, and often dying.

A relatively high concentration of copper is stored in the liver. This provides the principal reserve supply which is drawn on when the intake of copper is insufficient to counteract

that lost in secretions and excreta. Marston (1952) reported that only a small proportion of the copper in feedstuffs is absorbed by the animal and the major part of that ingested is excreted in the feces. Much of the copper that is excreted comes from copper-rich bile secretions. He felt that for ruminants the nature of the feed determined the efficiency with which the copper was absorbed and retained.

Hewetson et al., (1963) conducted tests on yearling cattle with low reserves of copper in the liver. The repeated subcutaneous injection of 120 mg of copper as glycinate failed to increase weight gain. They also injected pregnant cows with low copper reserves at three month intervals during pregnancy. This resulted in an increased level of copper in the liver and also increased the level in the blood above that of the untreated cows. The calves from these cows had higher levels of copper in the blood than from the untreated cows. However, they found no significant differences in birth weight or rate of gain.

Engel et al., (1964) did work with Holstein calves and found that a basal ration containing approximately four p.p.m. of copper resulted in a lower concentration of blood and liver copper than when copper sulphate was added to the ration to make the ration contain ten p.p.m. of copper. Supplementation of this ration with manganese, cobalt, zinc, and iron had no significant effect on liver copper. The workers observed no differences in growth, reproduction, milk production or copper content of body organs or muscle tissue between groups receiving

5 and 20 p.p.m. of copper. They also found liberal stores of copper in the livers of fetuses whose dams had received less than five p.p.m. of copper and had low concentrations of copper in the liver.

Cattle suffering from an insufficient amount of copper fail to have an appetite and become anemic as reported by Marston (1952). Milk production is then seriously decreased. Also, in mature cattle, the copper-deficiency syndrome frequently ends in heart failure, the result of atrophic lesions in the myocardium. Marston found the concentration of copper in the blood of cattle on normal copper intake rations was around one μg of copper per ml., while on a deficient ration, it was reported as low as 0.2 μg of copper per ml. Normal cattle have an average of approximately 200 μg of copper per gm of dried liver; but on a deficient diet, the amount of copper has been observed to be as low as two μg of copper per gram of dried liver.

Maynard and Loosli (1962) reported a copper deficiency disease of lambs called "sway back", which is characterized by nervous symptoms. They accomplished prevention of this disease by feeding copper to the pregnant ewes.

The concentration of copper in calves liver has been found to be five to ten times that found in livers of mature cattle. This must be Nature's way of supplying the calf with copper since it is a well known fact that bovine milk is very low in copper. The liver concentration of copper is also affected by dietary molybdenum and inorganic sulphate as previously mentioned

in this paper.

Underwood (1962) stated the minimum copper requirements for cattle are of limited value since there are interrelationships among dietary levels of copper, molybdenum, and sulphate. High levels of molybdenum intake often appear to cause a copper deficiency. High sulphate levels in turn seem to cause urinary molybdenum excretion. Underwood (1962) reported that in some areas it appeared the minimum copper requirements of cattle were approximately four p.p.m. He pointed out that copper deficiencies have been reported in England for cattle consuming feed of normal molybdenum and sulphate levels and 7-14 p.p.m. of copper. It becomes obvious, then, that it is very difficult to place an exact value for copper requirements for cattle. Maynard and Loosli (1962) listed 50 mg of copper as the daily copper requirement for cattle, while Cole (1962) estimated that cattle require 1.8-2.5 mg of copper per pound of dry matter consumed. The National Research Council (1963) felt the requirements of beef cattle fell in the range of 2-4 mg per pound dry feed; and that when the ration was low in molybdenum and sulphate, that probably no more than 1.8-2.3 mg per pound air dry feed (approximately 4-6 p.p.m.) was needed.

Copper, being like some other trace elements, can be present in feeds in toxic amounts. This frequently causes severe problems for sheep raisers, but the possibility of copper toxicity to cattle being fed normal rations seems extremely small. One case of copper toxicity was reported by Underwood (1962).

The steer died after receiving five gm of hydrated sulphate for 17 weeks. This would be approximately 550 p.p.m. of hydrated copper sulphate, assuming the steer was eating 20 pounds of feed per day. Todd and Thompson (1965) reported the death of two calves fed a ration containing up to 500 p.p.m. of copper. The calves died after receiving the copper for 20-21 weeks.

Duncan and Epps (1958), working in Louisiana, found the following values for copper content of Pinehill bluestem and Slender bluestem:

Species and Maturity Stage	Copper p.p.m.
Pinehill bluestem	
Early leaf	44.0
Full leaf	39.0
Mature green	22.5
Slender bluestem	
Early leaf	51.5
Full leaf	43.0
Mature green	32.5

Work done by Glendening et al., (1952) at Manhattan, Kansas, produced the following values for copper content of big and little bluestem:

Prairie Grass, Mostly Dry Bluestem, Collected in Winter	Copper p.p.m.
Average	97
Minimum	82
Maximum	112
Prairie Grass, Mostly Green Bluestem, Collected on Date Shown	
April 19	60

From the literature available it would appear that bluestem grass contains five to ten times the amount of copper required by cattle. This should be more than an ample amount even though cattle absorb very little of the copper they consume. The possibility of a copper deficiency is even more remote since cattle store large amounts of the copper they absorb, and there is very little danger of a copper toxicity since this has been reported only for cattle on rations containing five times that found in bluestem grass.

COBALT

Cobalt is one of the latest minerals to be added to the list of essential minerals for ruminants. This mineral was found to be necessary in the diet of ruminants for the prevention of progressive emaciation and anemia. Cobalt was termed "essential" after much work was done concerning a common disease, known by many different names, which occurred in grazing livestock in certain areas of the world. Maynard and Loosli (1962) indicated that the discovery of a lack of cobalt, resulting from a lack of cobalt in the soil and likewise in the forage grazed, was reported independently but simultaneously in 1935 by Filmer and Underwood and by Marston and Lines. All were Australian researchers attempting to find the cause of certain naturally occurring diseases of sheep and cattle known locally as "coast disease" and "wasting disease". Following this discovery, cobalt-deficient areas were discovered in other parts of the world according to Maynard and Loosli (1962). Areas were found in Florida, Michigan, Wisconsin, New Hampshire, New York, North Carolina, and western Canada. Even in Mexico, as reported by Gomez et al., (1963), a large valley there had not been used for cattle raising for 70 years because the animals grazing on it did not survive. Research revealed the cattle appeared normal after receiving a cobalt supplement.

Snapp and Neumann (1960) wrote, "Symptoms of cobalt deficiency, like those of many other mineral deficiencies, are general rather than specific. Severe deficiency results in reduced

feed intake, emaciation, weakness, and even death." They felt that the unrecognized borderline cases in which performance and feed conversion were reduced was probably of even greater importance. Maynard and Loosli's (1962) description of a cobalt deficiency was very similar to Snapp and Neumann's. They described it as resembling a general malnutrition in which the animals became listless, lost appetite and weight, became weak and anemic, finally resulting in death. Morrison (1959) reported that cattle suffering from a cobalt deficiency had a depraved appetite and seemed to have a desire to eat hair or gnaw on wood or bark. Davis (1951) stated that a partial deficiency of cobalt resulted in muscular weakness, lowered milk production, decreased calf crop, dry hair coat, and a slightly lowered resistance to parasitic infection. In addition, Davis stated, however, that stiffness did not occur as found with a copper deficiency. Gibbons (1963) included diarrhea with intermittent constipation as problems often encountered with cattle on cobalt-deficient diets. He pointed out that most cobalt-deficiencies are related to a disturbance of rumen function. Marston (1952) reported that calves born of cobalt-deficient cows were weak at birth and seldom lived longer than a week. Underwood (1962) stated that the appearance of a severe cobalt-deficient animal was very much like that of a starved animal, except the mucous membranes were blanched and the skin was generally pale and fragile. Also noted by Underwood was that milder forms of cobalt deficiencies appeared only during certain seasons of the year, but he could

give no reason for this occurrence. In addition, he reported that young growing calves were more susceptible than mature cattle to a lack of cobalt. Nearly all the workers reported that the only certain diagnosis of a cobalt deficiency was the quick response of the deficient animal to cobalt feeding. Reportedly, upon treatment with cobalt, the animals quickly regained their appetite, increased in weight, and regained their normal health and vigor.

Clanton et al., (1963), working at the Nebraska Station, conducted experiments on two ranches in the Sandhills of North Central Nebraska and at the Fort Robinson Research Station in North Western Nebraska to determine if cobalt supplements would increase gains of growing cattle. The workers found the use of a 20 gram cobalt bullet, placed in the reticulum for very slow dissolving, did not increase gains in calves and yearlings when tested during two years at three locations.

Chapman et al., (1963) working in Florida with yearling heifers administered eight mg of cobalt per animal daily to check for any increased gains. The animals were being grazed at the time of study. The cobalt treatment resulted in a higher blood hemoglobin and packed cell volume and a significantly higher copper and lower iron level in the liver. The treatment was reported to have had no effect on body weight changes.

Cobalt is especially important to the ruminant since it is required for the proper functioning of the rumen microorganisms which require cobalt to synthesize vitamin B_{12}. Vitamin B_{12}

is in turn needed by the body for the process of blood formation, according to Gibbons (1963). Gibbons listed lack of cobalt in the cobalt-microorganisms-B_{12} relationship as a possible explanation of anemia in the ruminant. Here it should be noted that according to Marston (1952) cobalt must be ingested by the ruminant since it was found to be of no value when injected into the blood stream. He found cobalt was absorbed above the duodenum.

Underwood (1962) explained that when an autopsy was run on an animal suffering from a severe cobalt deficiency, the body was often absent of fat. He stated that the liver was generally found to be fatty, the spleen hemosiderised, and hypoplasia of erythrogenic tissue in the bone marrow. A blood check before death revealed that both red cell numbers and blood hemoglobin levels were low.

Underwood (1962) reported the exact cobalt requirement of bovines was not known. He mentioned that daily supplements of 0.3-1.0 mg of cobalt have proved adequate for normal growth and health of cattle grazing pastures of severe cobalt deficiency. Underwood also reported a significant improvement in weight gains of dairy heifers when fed a cobalt supplement. The ration without the supplement was estimated to contain 1.2 mg of cobalt per day. The Merck Veterinary Manual (1961) stated natural forages containing less than 0.04 p.p.m. were usually considered deficient for cattle. They felt that feeding 5-15 mg of cobalt per day would cure a cobalt deficiency and as little as one mg would prevent its occurrence. This could be added to the feed,

or in range country it could be administered in the form of a "bullet". It was found, however, that occasionally the "bullet" was regurgitated and lost. Snapp and Neumann (1960) reported the cobalt requirement had been set by the National Research Council (1963) at 0.07-0.10 mg per 100 pounds body weight (approximately 0.03-0.05 mg per pound of feed or 0.07-0.11 p.p.m.). Gibbons (1963) stated that the requirement was less than one mg per day, while Morrison (1959) felt that 0.1 p.p.m. of cobalt would meet the cobalt requirement of the bovine.

It appeared that the literature was in relatively close agreement that the bovine needs approximately 0.07-0.12 p.p.m. of cobalt daily.

Duncan and Epps (1958) gave the following values for cobalt content of Pinehill bluestem and Slender bluestem grass:

Species and Maturity Stage	Cobalt (p.p.m.)
Pinehill bluestem	
Early leaf	0.54
Full leaf	0.13
Mature green	0.24
Slender bluestem	
Early leaf	0.50
Full leaf	0.36
Mature green	0.60

Glendening et al., (1952) obtained the following values for cobalt content of bluestem grass in North Central Kansas:

Prairie Grass, Mostly Dry Bluestem, Collected in Winter	Cobalt (p.p.m.)
Average	0.38
Minimum	0.26
Maximum	0.51
Prairie Grass, Mostly Green Bluestem, Collected on Dates Shown	
April 19	0.47
July 17	0.48

Based on the requirements given in recent literature, cattle on bluestem grass in North Central Kansas would not suffer from a cobalt deficiency.

IRON

Iron is truly considered a trace element, being required in very minute quantities by the body. Maynard and Loosli (1962) reported that the body contained only 0.004 per cent of iron; however this small amount is extremely important for the normal functioning of the body. Iron is best known as a constituent of the respiratory pigment hemoglobin, a blood constituent essential for the functioning of all parts of the body. Muscle contains the compound myoglobin according to Dukes (1955). In addition, he described hemoglobin as "a complex, iron-containing, conjugated protein composed of a pigment and a simple protein." This hemoglobin contained over 50 per cent of the body's iron supply. Dukes also reported that another form of iron other than hemoglobin occurred in the blood in the form of transport iron, which is available for hemoglobin synthesis and iron storage.

Maynard and Loosli (1962) felt that iron is cycled in the body. The red blood cells are continually broken down and replaced by the body. During this breaking-down process the hematin containing the iron is split off the hemoglobin molecule and is stored in the liver and spleen. This iron can be used over again with practically no loss. According to Dukes (1955) only a very small amount of iron is excreted from the body. These losses are replenished by drawing on the liver and spleen, which in turn are resupplied by absorption of iron from the digestive tract. This iron is absorbed primarily from the small

intestine. Maynard and Loosli (1962) felt that once the body stores are met, the adult animal needs very little iron in its ration except for productive purposes. Of course, this would not be true if the animal had a large blood loss or some other pathological condition. They stated that "the animal normally regulates iron absorption in accordance with its needs.". During pregnancy there is an increased absorption due to the needs of the developing fetus. Maynard and Loosli (1962) reported that it appeared the maintenance requirement must be increased two or three times to cover the needs of gestation. The newborn animal needs a relatively large supply of iron so that it can be drawn upon for blood formation during the suckling period when the primary feed is milk which is very low in iron. Milk is so low in iron, in fact, Dukes (1955) listed it as containing only 0.5 mg per liter. Underwood (1962) pointed out that calves on an all milk diet have long been known to develop an iron deficiency resulting in anemia. He reported that work done by Matrone and co-workers indicated that the minimum nutritional iron requirement of growing calves is not in excess of 30 mg per day. Rice and Nelms (1964), of Wyoming, experimented with treating beef calves with iron-dextran. The workers found that "calves injected with 1200 mg of elemental iron as iron-dextran per 100 pounds body weight at approximately two months of age were significantly ($P < .05$) heavier at weaning than calves not treated or calves treated at birth and again at two months of age. Calves injected at approximately two months of age were 42

pounds heavier at weaning than controls." The supplement resulted in increased blood hematocrit and hemoglobin levels at one month of age; however these differences were no longer evident at weaning.

Underwood (1962) stated that little is known about the iron requirement of cattle mainly because all ordinary rations of bovine generally contain an abundance of iron, thereby eliminating iron deficiency problems. He reported that no requirement could be made with any degree of confidence since there is virtually no knowledge of the absorptive efficiency of iron in the ruminant. Maynard and Loosli (1962) felt that it was extremely unlikely that a deficiency of iron ever occurs in a mature ruminant. The Merck Veterinary Manual (1961) stated that years ago iron deficiencies had been found; but since that time, it had been shown that copper and cobalt deficiencies had been involved in the original studies, and they questioned the existence of an iron deficiency in ruminants consuming natural feeds. Davis (1951), working at the Florida Agricultural Experiment Station, did, however, obtain beneficial results from the addition of 3-25 pounds red oxide of iron per 100 pounds of mineral mixture. The results were obtained from experiments run on cattle being pastured on light sandy soils. There seemed to be several logical explanations for these beneficial results. First these soils were probably leached thereby being low in iron content. Second there would be a possibility of a copper or cobalt deficiency in this area. And third the cattle had been troubled

with heavy insect infestations and parasitic infections and would need an above normal level of iron due to loss of blood caused by the insects and parasites. Smith et al., (1963) conducted an experiment in which steers on a high energy ration were injected intramuscularly at the beginning of the experiment and on the 119th day with ten cc. of an injectable iron compound containing 100 mg of iron per cc. The workers found that the injectable iron significantly depressed growth to a small degree. This experiment was the only piece of information found where iron had a known objectionable effect on cattle. It can be concluded from the preceeding discussion that the iron requirement for the mature bovine actually has not yet been determined. From work done it appears that cattle on normal rations receive a sufficient amount of iron.

Underwood (1962) listed pasture grasses as containing 100-200 p.p.m. of iron on the dry matter basis. Maynard and Loosli (1962) gave a value of 50-100 mg (110-220 p.p.m.) of iron per pound of roughage for cattle. Morrison (1959) gave three values for the iron content of bluestem grass: Active growth 0.024 per cent (240 p.p.m.), mature, 0.033 per cent (330 p.p.m.), mature and weathered 0.033 per cent (330 p.p.m.). Duncan and Epps (1958) gave the following values for iron content of Pinehill bluestem and Slender bluestem grasses:

Species and Maturity Stage	Iron (p.p.m.)
Pinehill bluestem	
Early leaf	132.0
Full leaf	100.0
Mature green	70.0
Slender bluestem	
Early leaf	186.0
Full leaf	134.0
Mature green	78.0

Glendening et al., (1952) obtained the following values for iron content of bluestem grass in North Central Kansas:

Prairie Grass, Mostly Dry Bluestem, Collected in Winter	Per cent Iron
Average	.040
Minimum	.034
Maximum	.047
Prairie Grass, Mostly Green Bluestem, Collected on Date Shown	
April 19	.047

The iron content values would be a range of 340-470 p.p.m. of iron.

From all available information concerning iron requirements and the iron, copper, and cobalt content of bluestem, it appears that iron presents no deficiency or toxicity problems for cattle grazing on bluestem grass.

IODINE

Iodine is required by the animal in extremely small quantities. Maynard and Loosli (1962) reported that it had been estimated the mature animal body contained less than 0.00004 per cent of iodine. Morrison (1959) stated that iodine was extremely important to the body for the production of thyroxin, an iodine-containing hormone, which is secreted by the thyroid gland in the neck. This hormone is necessary for all animals in regulating the rate of metabolism of the body; influencing growth; affecting other endocrine glands such as the gonads; and affecting body covering such as hair in the case of cattle. If sufficient iodine is not present in the diet, the thyroid will enlarge in an attempt to overcome the iodine deficiency, resulting in a goiter. In cattle a goiter condition was generally found by Morrison only in young calves. Underwood (1962) reported that iodine deficiency has been stated to be the most widespread of all mineral deficiencies in grazing stock.

The Merck Veterinary Manual (1961) gave the following as iodine deficiency symptoms: "big neck" or goiter in calves at birth; affected animals often bloated, having a reduced hair covering; and animals sometimes found to have an unusually thick skin. The length of pregnancy was usually prolonged, however many of the calves appeared premature at birth. Mortality at or soon after birth was common. The National Research Council (1963) stated that if these calves survived, the enlarged thyroid disappeared within a few weeks. Dukes (1955) reported a

lowered metabolism, mental sluggishness, lack of muscle tone, and impaired fertility in both sexes, in addition to some of those symptoms given by the Merck Veterinary Manual (1961).

Dukes (1955) reported that Fenger and Seidell found that external temperature appeared to be the most important factor involved in regulating the iodine content of the thyroids of cattle, sheep, and hogs. They found during winter increased size of the thyroid led to a lower per cent of iodine content. They also found that during cold winter temperatures, increased secretions, needed to maintain body temperature, was another factor affecting thyroid iodine content. Underwood (1962) wrote that concentration of iodine in the thyroid was related to the iodine intake, to the activity of the gland, and to the age of the animal, but not the sex of the animal. He stated the normal thyroid of a mammal contained 0.2-0.5 per cent of iodine on a dry matter basis. The iodine was stored in largest quantity in the thyroid in a protein form called thyroglobulin.

Several areas in the United States are known to be iodine-deficient or "goiter areas". These areas are primarily in the Northwest and Great Lakes regions. Dukes (1955) reported that deficient areas had also been found in Virginia, West Virginia, Pennsylvania, and Missouri. It was noted that all these areas had a deficiency of iodine in the soil and were generally limestone areas. Some workers felt that an excess of lime could have been a factor leading to the iodine deficiency.

Cabbage, soybeans, and turnips often cause goiter in

livestock according to the Merck Veterinary Manual (1961). These feeds contain substances which inhibit thyroxine production. But it was reported that when raw soybeans are processed, inhibition of thyroxine production is partially destroyed.

Iodine is unique among the trace elements since the level of iodine in the ration greatly affects the level of iodine in the milk. Underwood (1962) felt the level of iodine in the milk was influenced by the stage of lactation, the season of the year, and the geographical region. However, the season and the geographical location both primarily influence the level of iodine intake.

Underwood (1962) believed that iodine occurs in feed in the form of an inorganic iodide and is absorbed throughout the gastrointestinal tract in this iodide form. A very minute amount of iodide is lost from the body in the form of sweat, although the main pathway of excretion of iodide is through the kidneys.

Underwood (1962) stated that metabolism of iodine could be considered in relation to the movements of the element into and out of the body's "inside pool". The total iodide pool was said to consist of the iodide present in the extracellular space, together with the red blood cells and certain areas of selective concentration such as the thyroid, salivary, and gastric glands and dense connective tissue. This iodide was replenished continuously from two sources, one being from the diet eaten by the animal and the other from the breakdown of iodine hormones produced by the thyroid.

To date there seems to have been no quantitative studies done to determine the exact iodine requirement of the bovine. The National Research Council (1963) estimated the requirement of a 1000 pound dairy cow, producing 40 pounds of milk per day, to be 400 to 800 mg (880-1760 p.p.m.) of iodine per day. They found the use of iodized salt containing 0.01 per cent stabilized potassium iodide (0.0076 per cent of iodine) prevented iodine deficiency symptoms. This same recommendation was made by Cole (1962) and Underwood (1962).

Underwood (1962) described the iodine content of plants as highly variable. The species of plant, the soil type, seasonal and climatic conditions all played a part in the iodine content of the forage. He stated that results obtained from a great number of analyses carried out in different parts of the world revealed an average iodine content of 300-500 µg per kg of dry roughage.

It appeared from the literature that of all feedstuffs for cattle, grass is one of the richest sources of iodine. Since cattle require relatively small amounts of iodine per day, it would appear that cattle grazing on bluestem grass receive an ample supply in their diet. This would certainly seem to be true, unless the high amount of limestone in the area would be a contributing factor leading to an iodine deficiency.

MANGANESE

Morrison (1959) reported that trace amounts of manganese were essential for all plants and animals. Maynard and Loosli (1962) found the largest amount of manganese occurred in the liver, but appreciable amounts were found in other organs along with the skin, muscle, and bones. Underwood (1962) stated that manganese was more concentrated in the mitochondria than in the cytoplasm or other parts of the cell. Underwood also reported that varying the levels of manganese in the diet did not change the manganese levels in the liver to any significant extent as found in some of the other trace minerals. New born calves were found to have an extremely low level of manganese in the liver. Underwood pointed out that this was surprising since cow's milk is relatively deficient in manganese, containing an average of 0.03 mg of manganese per liter. However, cow's colostrum was reported to have been five times richer in manganese than ordinary milk. It was also found that the level of manganese in milk could be raised by feeding additional manganese.

Dukes (1955) reported that manganese was eliminated by the colon, in the bile, and in the urine. He reported that the functions of manganese in the body were poorly understood, but that evidence indicated that it was necessary for normal bone formation, growth, and reproduction. Feaster (1963) found that manganese was essential in the prevention of nervous disorders of animals. Dukes (1955) also suggested that manganese may have been involved in tissue respiration and that manganese was the

activating ion of arginase.

According to Underwood (1962) there was no definite data available on the manganese content of blood. Work done was described as meager and discordant. A level of 2-8 µg per 100 ml of whole blood was given for calves. Underwood also reported an increase in serum manganese from a mean of one to a mean of 150 µg per 100 ml of blood showed-up in cows when turned out to pasture in certain areas of England. This would indicate that the level of manganese in the feed is highly correlated to the manganese level in the blood. Rojas and Dyer (1964) listed a value of 2.78 mg per 100 ml for cows receiving a ration ample in manganese. Rojas et al., (1965) found that manganese-deficient rations fed to calves resulted in a lowered manganese blood level. In an experiment conducted by Gessert et al., (1952), working with Holstein cows, it was found that by supplementing a control ration, which was apparently adequate in manganese, with a manganese supplement that little or no change in the manganese blood concentrations occurred.

Manganese deficiency symptoms for cattle have not been clearly described since most practical rations contain a higher level of manganese than most workers feel is required. Bentley and Phillips (1950) found that heifers raised on a ration containing less than 10 p.p.m. of manganese were slower to exhibit estrus and were slightly but consistently slower to conceive upon breeding. They found the ovaries, taken from heifers fed this ration, contained an exceptionally low manganese concentra-

tion, despite the presence of normal levels of manganese in the other body tissues examined. The workers also found that low dietary manganese caused abnormal structural changes in the livers of cattle. Rojas et al., (1965) at Washington State University, conducted an experiment with four-year-old Hereford cows in an attempt to determine the major changes that take place in beef cattle associated with a manganese deficiency. The rations fed all these cows were the same except the control ration was fortified with manganese. The daily intakes of manganese were: 182.8 mg (25.1 p.p.m.) for the controls and 114.7 mg (15.8 p.p.m.) for the low manganese group. The results showed the manganese deficiency did not affect weight gain, feed consumption or feed efficiency. However, breeding efficiency was impaired. The controls required an average of two services per pregnancy in comparison with the manganese-deficient cows which required an average of four services. The manganese-deficient cows continued to cycle regularly; however the reproductive performance was believed to be altered due to a delayed ovulation. All calves from the deficient cows were deformed. The dams fed deficient manganese rations had enlarged joints, stiffness, twisted legs, and a general weakness. The bones of the deficient calves contained less manganese and had a reduced breaking strength. Blood manganese and testicular manganese content were both lowered in the manganese-deficient calves.

Workers have tried to find methods of determining marginal manganese deficiencies but have met with little success.

Chauvaux et al., (1965) found that a manganese deficiency could not be diagnosed on the basis of the manganese content of hairs.

There were early reports that manganese was of value in the prevention and cure of brucellosis. Davis (1951) at Florida State University found no difference in the incidence of brucellosis between herds receiving manganese and those not receiving manganese. Gossert et al., (1952) also investigated the possible correlation between brucellosis and the manganese concentration in the livers of cattle. These workers found approximately the same concentration of manganese and other trace minerals in the livers of cattle with brucellosis as those which had not been exposed to the Brucella abortus organism.

The National Research Council (1963) stated the manganese requirement of beef cattle was uncertain but appeared to be as low as 2.7-4.5 mg (6-10 p.p.m.) per pound of dry feed. Gibbons (1963) stated that it had been reported by the California Experiment Station that beef heifers fed a limited amount of a high energy ration containing six p.p.m. (2.7 mg per pound) of manganese produced normal calves. Morrison (1959) reported that growth rate and sexual maturity of dairy calves was delayed on a ration containing only 6-8 p.p.m. manganese (2.7-3.6 mg per pound) dry feed. Investigations by Rojas and Dyer (1965) indicated that the requirement of manganese in cows was in excess of 16 p.p.m. rather then 6-10 p.p.m. given by the National Research Council. These Washington State workers (Rojas and Dyer) felt the true manganese requirement was approximately 20 p.p.m. (9.0

mg per pound) air dry feed. Bentley and Phillips (1950) felt that 20 p.p.m. (9.0 mg per pound) manganese dry feed in the ration was adequate for the dairy cow.

Fain et al., (1952) conducted an investigation in an attempt to determine the effect of manganese in different amounts on the glucose, calcium, manganese, iron, and potassium levels in the blood of cattle and to try to determine if manganese was a factor in causing "wheat poisoning", a disease often found in cattle grazing lush green wheat. The ration was supplemented with manganese at levels of 75, 100, 150, and 200 p.p.m. of manganese. The manganese in the blood serum exhibited a downward trend during the period when the cattle were receiving 100 p.p.m. manganese; but the values increased sharply when the cattle began receiving 150 p.p.m. manganese, and they remained at approximately normal concentration of manganese thereafter. The depression of manganese was not clearly understood; however it was thought that a possible disturbance of manganese metabolism might occur at a manganese concentration of approximately 100 p.p.m. to which cattle might be subjected in grazing wheat.

Robinson et al., (1960) experimented with excess dietary manganese and feed lot performance of beef cattle. Normal rations were supplemented with 0, 25, 500, and 1000 p.p.m. manganese. Daily gains were 1.63, 1.51, 1.57, and 1.58 pounds per day respectively, and feed efficiency was 9.8, 10.6, 10.2, and 10.2 respectively. Also noticed was that excess manganese decreased iron absorption and reduced fiber digestibility. Tissue

analysis showed an increase of manganese storage and a depletion of iron. The workers felt that the data indicated the effects of excess manganese to be greater on function than on structure in tissues and organs studied.

Manganese is widely distributed in ordinary feeds and generally the small amount present more than meets the requirements of the bovine. Morrison (1959) listed the following values for the manganese content of bluestem grass:

	Manganese	
	mg per #	p.p.m.
Bluestem pasture, active growth	12.9	28.7
Bluestem pasture, mature	8.5	19.0
Bluestem pasture, mature and weathered	18.0	40.0

Duncan and Epps (1958) listed the following values for the manganese content of Pinehill bluestem and Slender bluestem:

Species and Maturity Stage	Manganese (p.p.m.)
Pinehill bluestem	
Early leaf	376
Full leaf	40
Mature green	102
Slender bluestem	
Early leaf	492
Full leaf	560
Mature green	486

Glendening et al., (1952) found the following values for bluestem grass in North Central Kansas:

Prairie Grass, Mostly Dry Bluestem, Collected in Winter	Manganese (p.p.m.)
Average	71
Minimum	61
Maximum	82
Prairie Grass, Mostly Green Bluestem, Collected on Dates Shown	
April 19	88

It was readily apparent that the manganese content of the grass was far in excess of that believed necessary for a normal manganese requirement; and because of this it was suggested that grazing on grass possibly could have toxic effects to cattle.

In summary, it appeared from the literature that the manganese requirement of beef cattle would be met if the ration contained as much as 10-20 p.p.m. of manganese (4.5-9 mg per pound) dry fed. Also from the preceding literature it would certainly appear that bluestem grass contains more than enough manganese to meet the requirements of cattle. However, the levels of manganese for bluestem in the Manhattan, Kansas, area did not appear to be high enough to cause any serious antagonizing effects on the other minerals present.

SUPPLEMENTING ROUGHAGE RATIONS WITH TRACE MINERAL MIXTURES

Gossett and Rigge (1956) of Texas found no benefit from adding trace minerals to a growing ration consisting of poor quality prairie hay, two pounds of cottonseed meal, and four pounds of ground milo grain. Tillman et al., (1954) obtained no response from the addition of a complete mineral mixture to sheep rations made of low quality prairie hay.

Nelson et al., (1951) conducted experiments which showed a decrease in weight gain and a less thrifty appearance by feeding trace minerals to heifers on a roughage wintering ration. However, during the summer these heifers out-gained the non-trace mineral fed heifers. The data, therefore, indicated that trace minerals were of no benefit or harm to the animals.

Tests by Nelson et al., (1952) working with beef heifers and Nelson (1954) and (1955) working with stocker cows which were grazed in Southeast Oklahoma and fed a protein supplement showed no improvement in growth or general appearance of cattle fed a trace mineral mixture which included iron, cobalt, copper, iodine, and manganese.

Totusek and Gallup (1956) attempting to improve reproductive performance in cattle in Southeast Oklahoma obtained no significant differences in weight and appearance of cows or weaning weight of calves by the addition of trace minerals to the diet of the grazing stock.

Tillman and Sirny (1959), in an attempt to give a more

critical test, fed weathered range grass to cattle in a drylot. Results showed that when fed a cottonseed meal supplement, no significant difference in weight gain was obtained by feeding trace minerals. However, the animals did respond to the trace minerals by displaying a glossier haircoat and less dandruff than the control animals.

Smith and Cox (1952, 1953) fed a trace mineralized salt containing iodine, copper, cobalt, iron, and manganese to steer calves on a wintering ration. The experiment revealed no benefit from adding the trace mineral elements. Smith et al., (1954, 1955) repeated these trials and observations. Koch et al., (1960) reported no significant differences in gain of cattle on bluestem grass between those receiving trace minerals and those not receiving trace minerals. McCartor et al., (1961) fed an all roughage ration of silage and prairie hay to steers and were unable to show any effect on daily gain by the use of trace mineral feeding.

Dowe et al., (1955, 1956) found that steers on a high roughage ration of brome grass hay and limited corn and soybean meal did not give additional response when the trace minerals cobalt, copper, manganese, iron, iodine, and zinc were fed in a salt mixture.

Plumlee et al., (1953) fed identical twins corn cobs with added trace minerals. Results showed that trace minerals depressed the appetite and therefore controlled the intake of feed.

SUMMARY

The purpose of this report was to summarize some of the results obtained from experiments conducted by others concerning the trace mineral requirements of cattle. An attempt was made to try to determine if cattle grazing native bluestem grass would be deficient in any of these mineral elements: zinc, molybdenum, copper, iron, iodine, manganese, and cobalt.

From the work of Underwood (1962), Maynard and Loosli (1962), Ott (1965), Miller (1963), and others, it can be seen that no definite zinc requirements for cattle has been established. Work done shows that the requirement appears to fall in the range of 20-30 p.p.m. of zinc on the dry matter basis. Due to the interference of zinc function by calcium, which is relatively high in most bluestem grass, it would appear that the zinc requirement for cattle on bluestem grass would be at least 30 p.p.m. of zinc.

Underwood (1962) classed molybdenum as an essential trace mineral element since beneficial response from molybdenum has been obtained when ruminants were fed a semipurified diet containing 0.36 p.p.m. of molybdenum. However, molybdenum receives much more attention from the standpoint of toxicity rather than from deficiency. Work revealed there is a possibility of molybdenum toxicity to cattle when the feed contains in excess of approximately 10 p.p.m. of molybdenum on the dry matter basis. Higher levels of molybdenum can be present in the ration without

harmful effects if sufficient copper and sulfate are present since there is a three-way interrelationship among copper, sulfate, and molybdenum. High levels of copper and sulfate reduce molybdenum retention and thus reduce the chances of molybdenum toxicity.

Work by Glendening et al., (1953) on molybdenum and copper content of bluestem grass makes it appear that sufficient amounts of molybdenum are present and that the possibilities of molybdenum toxicity are extremely remote.

Underwood (1962) stated the minimum copper requirement for cattle is of limited value due to the interrelationship that exists among copper, molybdenum, and sulfate. High levels of molybdenum appear to cause a copper deficiency. It appeared from the literature that the requirement for copper by beef cattle falls in the range of four-six p.p.m. A copper deficiency appeared to be of no problem in the Kansas bluestem area since this grass contained approximately 100 p.p.m. of copper.

The literature was in relatively close agreement as to the daily cobalt needs of the bovine. Work revealed the bovine needs approximately 0.07-0.12 p.p.m. of cobalt daily. Based on this requirement, cattle on bluestem grass in North Central Kansas should not suffer from a cobalt deficiency since grass in that area was found to contain from 0.26-0.51 p.p.m. of cobalt.

Underwood reported the exact iron requirement of cattle was unknown, but it was believed that all ordinary rations of

cattle contain an abundance of iron, thereby eliminating iron deficiency problems.

From the literature, it appeared that iodine deficiencies are probably the most widespread of trace mineral deficiencies of grazing stock. Iodine-deficient areas occur primarily in the Northwest and Great Lakes regions. However, other areas have been reported in the United States as being iodine deficient. It is interesting to note that most of these iodine-deficient areas are also limestone areas. It was thought that limestone might possibly be a factor leading to the iodine deficiency. Since grass has been found to be one of the richest sources of iodine, it would seem that cattle grazing native bluestem pasture would not suffer from an iodine deficiency unless possibly the high calcium content of the soil interferes with the absorption of iodine by plants.

There is considerable disagreement in the literature as to the manganese requirements of beef cattle. The National Research Council (1963) felt the manganese requirement was approximately 6-10 p.p.m., while Rojas and Dyer (1965) thought it was approximately 20 p.p.m. It thus appears the manganese requirement falls in the range of 10-20 p.p.m. This range of 10-20 p.p.m. appears to be easily met in the Kansas bluestem area since work revealed that bluestem in this region contained 60-90 p.p.m. manganese, a level which appeared to have no antagonistic effect on the bovine.

In the past two decades considerable work has been done in

an attempt to determine the value of added trace minerals for cattle on roughage rations. The results obtained in the Central United States were almost in complete agreement that there was no benefit to supplementing roughage rations with trace minerals.

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THE VALUE OF TRACE MINERALS FOR CATTLE GRAZING BLUESTEM GRASS

by

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The literature contains much conflicting information as to the exact requirements of the trace mineral elements needed by the bovine.

No definite zinc requirement for cattle has been established. Research revealed that the zinc requirement appeared to fall in the range of 20-30 p.p.m. of zinc on the dry basis. Due to the interference of zinc function by calcium, which is relatively high in most bluestem grass, it would appear that the zinc requirement for cattle on bluestem grass would be at least 30 p.p.m. of zinc.

Molybdenum has received much more attention from the standpoint of toxicity than from a deficiency standpoint. Molybdenum toxicity was possible when the feed contained in excess of ten p.p.m. of molybdenum. High levels of copper and sulfate reduced the chance of molybdenum toxicity. The possibility of a molybdenum toxicity problem for cattle grazing in the North Central Kansas area seems extremely remote.

The literature contained no definite copper requirement for cattle; however it appeared the copper requirement was in the range of four-six p.p.m. of copper. A copper deficiency appears to be no problem in the Kansas bluestem area since this grass contains approximately 100 p.p.m. of copper.

The bovine appeared to need approximately 0.07-0.12 p.p.m. of cobalt daily. This seems to be met by cattle on bluestem grass which was found to contain 0.26-0.51 p.p.m. of cobalt.

The exact iron requirement of cattle is unknown but it is

believed that all ordinary rations of cattle contain an abundance of iron, thereby eliminating iron deficiency problems.

Iodine deficiencies are probably the most widespread of trace mineral deficiencies of grazing stock. Iodine-deficient areas occur primarily in the Northwest and Great Lakes regions. Other areas high in limestone content have been reported as being iodine-deficient areas. Since grass has been found to be one of the richest sources of iodine, it would seem that cattle grazing native bluestem pasture would not suffer from an iodine deficiency unless possibly the high calcium content of the soil interfered with the absorption of iodine by plants.

It appeared the manganese requirement falls in the range of 10-20 p.p.m. which is easily met by cattle on bluestem grass since it has been found to contain from 60-90 p.p.m. of manganese.

No beneficial results have been obtained from supplementing roughage rations with trace mineral mixtures in the Central United States.